9/16/03

File 155:MEDLINE(R) 1966-2003/Sep W1
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***File 155: Medline has been reloaded and accession numbers have** changed. Please see HELP NEWS 155.

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      Set  Items  Description
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?e c3f

Ref   Items  Index-term
E1        1  C3E1
E2        1  C3E2
E3       79 *C3F
E4        1  C3FEB6
E5        1  C3FEB6F
E6        1  C3FEB6F1
E7        2  C3FF
E8        1  C3FO
E9        1  C3FRARE
E10       1  C3FRG
E11       1  C3FROM
E12       8  C3FS

          Enter P or PAGE for more
?s e3-e12
              79  C3F
               1  C3FEB6
               1  C3FEB6F
               1  C3FEB6F1
               2  C3FF
               1  C3FO
               1  C3FRARE
               1  C3FRG
               1  C3FROM
               8  C3FS
      S1      87  E3-E12
?s s1 and (peptide? or polypeptide? or fragment? or epitope? or portion? or 15 or 16)
              87  S1
          330372  PEPTIDE?
           81227  POLYPEPTIDE?
          245942  FRAGMENT?
           73960  EPITOPE?
           86105  PORTION?
          611431  15
          397580  16
      S2      17  S1 AND (PEPTIDE? OR POLYPEPTIDE? OR FRAGMENT? OR EPITOPE?
                  OR PORTION? OR 15 OR 16)
?t s2/9/all
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2/9/14
DIALOG(R)File 155:MEDLINE(R)

04891723 85198284 PMID: 6534316

Polymorphism of C3 component of complement in the Polish population. I. Population and family studies.

Manczak M

Archivum immunologiae et therapiae experimentalis (POLAND) 1984, 32 (4) p421-30, ISSN 0004-069X Journal Code: 0114365
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

In a sample of the Polish population numbering 4741 subjects, the three common types C3S, **C3F** and **C3FS** and **15** phenotype variants were found with frequencies 0.0046. The frequencies of C3S and **C3F** genes determining the common types were 0.8227 and 0.1750, respectively. Examination of 40 newborns and their mothers has revealed that C3 types are formed during the fetal life. The results of studies on 76 families with 157 children and 2332 mother-child pairs have confirmed that 3C3 types are determined by a single genetic locus in which codominant autosomal alleles are situated.

Tags: Comparative Study; Female; Human; Male
Descriptors: *Complement 3--genetics--GE; *Polymorphism (Genetics); Adult ; Alleles; Gene Frequency; Genes, Dominant; Infant, Newborn; Poland
CAS Registry No.: 0 (Complement 3)
Record Date Created: 19850606
Record Date Completed: 19850606

04660019 84303653 PMID: 6382974

Complement derived factors and prostacyclin formation by rabbit isolated peritoneum and cultured mesothelial cells.

Bult H; Coene M C; Rampart M; Herman A G

Agents and actions. Supplements (SWITZERLAND) 1984, 14 p237-47, ISSN 0379-0363 Journal Code: 7801014
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Activation of rabbit or human serum complement led to the generation of factors which stimulated prostacyclin biosynthesis by isolated rabbit peritoneal tissue. Their formation was proportional to the degree of complement activation, measured as consumption of total hemolytic activity or immunoreactive C3. The stimulation of prostacyclin biosynthesis was mimicked by **fragments** obtained by trypsinisation of C3 (**C3f**) and C5 (C5f). Peritoneal macrophages, which could stimulate peritoneal prostacyclin biosynthesis through release of chemical mediators in response to **C3f** or C5f, were not essential, since the **C3f** and C5f also stimulated prostacyclin biosynthesis in monolayers of cultured mesothelial cells. Of the putative mediators, platelet activating factor (PAF) was inactive as a stimulator of peritoneal PGI2 biosynthesis. The finding that activated complement components stimulate prostacyclin biosynthesis forms an explanation for the endotoxin-induced rise in rabbit arterial blood levels of prostacyclin and may have wider implications for the understanding of inflammatory reactions.

Tags: Animal; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.
Descriptors: *Complement 3--pharmacology--PD; *Complement 5--pharmacology --PD; *Epoprostenol--biosynthesis--BI; *Peritoneum--metabolism--ME; Blood --metabolism--ME; Cells, Cultured; Complement Activation--drug effects--DE; Epithelial Cells; Epithelium--metabolism--ME; Hemolysis--drug effects--DE; Platelet Activating Factor--pharmacology--PD; Rabbits
CAS Registry No.: 0 (Complement 3); 0 (Complement 5); 0 (Platelet Activating Factor); 35121-78-9 (Epoprostenol)
Record Date Created: 19841004
Record Date Completed: 19841004

04494343 84136999 PMID: 6607952

Physiologic inactivation of fluid phase C3b: isolation and structural analysis of C3c, C3d,g (alpha 2D), and C3g.

Davis A E; Harrison R A; Lachmann P J

Journal of immunology (Baltimore, Md. - 1950) (UNITED STATES) Apr 1984, 132 (4) p1960-6, ISSN 0022-1767 Journal Code: 2985117R

Contract/Grant No.: AI 17896; AI; NIAID; AM 16392; AM; NIADDK; RR 128; RR ; NCRR

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: AIM; INDEX MEDICUS

The **fragments** that result from the inactivation of C3b have not been completely characterized. Initial inactivation is catalyzed by the protease factor I, which, in the presence of its cofactor (factor H), cleaves two **peptide** bonds in the alpha'-chain of C3b. This results in the release of a small **peptide** (**C3f** , Mr 3000) from iC3b, which consists of the C3 beta chain covalently bonded to two alpha'-chain-derived **peptides** (Mr 68,000 and Mr 43,000). Surface-bound iC3b is cleaved at a third site by factor I to produce C3c and C3d,g (or alpha 2D). The factor I cofactor for this cleavage is the C3b receptor that is present on erythrocyte and leukocyte membranes. This report describes the isolation and initial structural characterization of C3c and C3d,g generated in whole blood after complement activation with cobra venom factor. These **fragments** were compared with the C3 **fragments** isolated from the serum and plasma of a patient with complement activation in vivo. The **fragments** were isolated with two solid phase monoclonal antibodies, one of which recognizes a determinant on C3g (clone 9) and one of which recognizes a determinant on C3c (clone 4). C3c isolated from normal blood showed three **polypeptides** that had apparent m.w. of 75,000, 43,000, and 27,000. The C3d,g consisted of a single **polypeptide** chain with a m.w. of 40,000. Amino terminal sequence analysis showed that the Mr 27,000 **peptide** from C3c is derived from the amino terminal **portion** of the alpha'-chain of C3b, whereas the Mr 43,000 **peptide** is derived from the carboxy terminus of the same chain. Amino terminal sequence analysis showed also that C3g is derived from the amino terminus of C3d,g. The C3 **fragments** isolated from a patient with partial lipodystrophy, nephritic factor activity, low serum C3 levels, and circulating C3 cleavage products showed a more complicated pattern on SDS-PAGE. The **fragment** isolated with clone 9 had an apparent m.w. of 40,000, identical to C3d,g generated in vitro, and it had the same amino terminal sequence as C3d,g generated in vitro. The eluate from insolubilized clone 4, however, showed prominent bands with Mr of 75,000, 56,000, 43,000, and 27,000, together with a triple-banded pattern at 68,000 and a minor band at 80,000. This eluate thus appears to contain C3c, and iC3b or an iC3b-like product. The origin of the Mr 56,000 and Mr 80,000 **peptides** have not yet been determined. These studies, with previous data, definitively order the C3c and C3d,g **peptides** in the alpha-chain of C3.(ABSTRACT TRUNCATED AT 400 WORDS)

Tags: Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Descriptors: *Complement 3--metabolism--ME; *Complement 3b Inactivators; Amino Acid Sequence; Cobra Venoms--pharmacology--PD; Complement 3--analysis --AN; Complement 3--biosynthesis--BI; Complement 3 --isolation and purification--IP; Complement 3c; Complement 3d; Lipodystrophy--immunology --IM; Molecular Weight; **Peptide Fragments** --analysis--AN; **Peptide Fragments** --biosynthesis--BI; **Peptide Fragments** --isolation and purification--IP

CAS Registry No.: 0 (Cobra Venoms); 0 (Complement 3); 0 (Complement 3b Inactivators); 0 (Peptide Fragments); 0 (cobra venom factor); 0 (complement 3g); 80295-44-9 (Complement 3c); 80295-45-0 (Complement 3d)

Record Date Created: 19840424

Record Date Completed: 19840424

05757008 88110500 PMID: 3338271

Structure of C3f , a small peptide specifically released during inactivation of the third component of complement.

Harrison R A; Farries T C; Northrop F D; Lachmann P J; Davis A E
MITI Unit, MRC Centre, Cambridge, UK.

Complement (Basel, Switzerland) (SWITZERLAND) 1988, 5 (1) p27-32, ISSN 0253-5076 Journal Code: 8409977
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

C3f , a **peptide** presumed to be generated by the combined actions of factors I and H on fluid-phase C3b, has been isolated and sequenced. The **peptide** is 17 residues long and has a molecular weight of 1,847 daltons. The amino-terminal sequence is, with the exception of a single residue, identical to that deduced for the 46-kilodalton **polypeptide** seen transiently in the generation of iC3b from C3b, and is in full agreement with the sequence deduced from cDNA analysis. In addition, high-pressure liquid chromatography of the digestion of C3b by factor I has shown that **C3f** is the sole **peptide** released during iC3b generation.

Tags: Human
Descriptors: *Complement 3--genetics--GE; Amino Acid Sequence; Complement 3--isolation and purification--IP; Complement 3b--metabolism--ME; DNA --genetics--GE; Molecular Weight
CAS Registry No.: 0 (Complement 3); 0 (complement 3f); 80295-43-8 (Complement 3b); 9007-49-2 (DNA)
Record Date Created: 19880311
Record Date Completed: 19880311

05712264 88065543 PMID: 2446123

Localization and functional significance of a polymorphic determinant in the third component of human complement.

Behrendt N; Hansen O C; Ploug M; Barkholt V; Koch C
Institute of Biochemical Genetics, University of Copenhagen, Denmark.

Molecular immunology (ENGLAND) Oct 1987, 24 (10) p1097-103, ISSN 0161-5890 Journal Code: 7905289
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

A polymorphic **epitope** in the third component of human complement was studied. This allotypic system is distinct from the electrophoretically determined C3 S/F polymorphism and is defined by the recognition of one allotype by a monoclonal antibody. Allotypic protein variants, **C3F** + (reactive with this antibody) and C3S- (non-reactive with the antibody), were purified. Deglycosylation studies and N-terminal sequencing of CNBr **fragments**, reactive with the antibody, revealed that the polymorphic **epitope** was present in a beta chain **fragment** of mol. wt 20,000. In the intact C3 molecule, this **fragment** is situated with N-terminus at residue No. 202, using the numbering of the cDNA derived amino acid sequence of human prepro C3. Addition of Fab **fragments** from the alloselective antibody preferentially inhibited the activity of **C3F** + in a haemolytic assay which is selective for the C3 activity in the alternative complement pathway.

Tags: Human; Support, Non-U.S. Gov't
Descriptors: Complement 3--immunology--IM; * **Epitopes** --analysis--AN; *Polymorphism (Genetics); Amino Acid Sequence; Antibodies, Monoclonal --immunology--IM; Complement 3--antagonists and inhibitors--AI; Complement 3--genetics--GE; Cyanogen Bromide; Electrophoresis, Polyacrylamide Gel; **Peptide Fragments** --immunology--IM
CAS Registry No.: 0 (Antibodies, Monoclonal); 0 (Complement 3); 0 (Epitopes); 0 (Peptide Fragments); 506-68-3 (Cyanogen Bromide)
Record Date Created: 19880104
Record Date Completed: 19880104

6456951 90081725 PMID: 2531841

Factor C3f **is a spasmogenic** fragment **released from C3b by factors I and H: the heptadeca-** peptide C3f **was synthesized and characterized.**

Ganu V S; Muller-Eberhard H J; Hugli T E

Pharmaceuticals Division, CIBA-GEIGY Corporation, Summit, NJ 07901.

Molecular immunology (ENGLAND) Oct 1989, 26 (10) p939-48, ISSN 0161-5890 Journal Code: 7905289

Contract/Grant No.: AI 17354; AI; NIAID; HL 07195; HL; NHLBI; HL 25658; HL; NHLBI; +

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

C3f , a heptadeca- **peptide** having the amino acid sequence of NH2-Ser-Ser-Lys-Ile-Thr-His-Arg-Ile-His-Trp-Glu-Ser-Ala-Ser-Leu-Leu-Arg-COOH, is liberated during the catabolic degradation of C3b in serum. The amino acid sequence of **C3f** is known both from the cDNA-derived structure of C3 and from protein analysis after isolation of the natural factor. **C3f** was synthesized by solid phase **peptide** synthesis. Both natural and synthetic **C3f** had identical retention times by RP-18 high performance liquid chromatography (HPLC) analysis and the respective amino acid compositions agreed with the expected theoretical values. **C3f** , but not des-Arg- **C3f** , was weakly spasmogenic inducing contraction of guinea pig ileum at a level of 5-10 x 10(-6) M. Since **C3f** and C3a were cross-tachyphylactic, it was concluded that these two spasmogens compete for the same receptors. Both **C3f** and des-Arg- **C3f** at concns of 1-4 x 10(-4) M enhanced vascular permeability in guinea pig skin. These observations further suggest that **C3f** functionally resembles C3a anaphylatoxin. Formation of **C3f** in human serum following CVF activation of C3 could be demonstrated by radioimmunoassay (RIA). Digestion of **C3f** with purified human serum carboxypeptidase N produced **C3f** -desArg. These observations suggest that when serum complement protein C3 undergoes conversion to C3b, further degradation by Factors H and I readily generates **C3f** . **C3f** is a weak spasmogen that functions like C3a anaphylatoxin and **C3f** -desArg is a major metabolite in serum.

Tags: Animal; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Descriptors: *Complement 3--physiology--PH; *Complement 3b--metabolism --ME; *Complement 3b Inactivators--pharmacology--PD; *Serine Endopeptidases --pharmacology--PD; Amino Acid Sequence; Capillary Permeability;

7728592 93183869 PMID: 8443150

Studies on the structure of complement C3 and the stability of C3 derived phagocytic ligands C3b/iC3b in SJL/J and BALB/c mice.

Lynch D M; Kay P H; Papadimitriou J M; Grounds M D

Department of Pathology, University of Western Australia, Nedlands.

European journal of immunogenetics - official journal of the British Society for Histocompatibility and Immunogenetics (ENGLAND) Feb 1993, 20 (1) p1-9, ISSN 0960-7420 Journal Code: 9106962

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Female SJL/J mice are more susceptible to development of experimental autoimmune myositis than most other mouse strains. Since complement has been implicated in the pathogenesis of inflammatory muscle disease in humans, quantitative and qualitative studies of complement C3 were undertaken in SJL/J and BALB/c mice to determine whether complement may influence disease susceptibility in SJL/J mice. In accordance with previous studies, mature male and female BALB/c mice were shown to have similar serum C3 concentrations. However, differences were found between mature male and female SJL/J mice. Male SJL/J mice have significantly higher serum C3 concentrations than SJL/J females and both sexes of BALB/c mice suggesting that serum C3 concentration may be variably influenced by sex in some mouse strains. Qualitatively, SJL/J mice were shown to have a different allotypic form of C3 (**C3F**) compared to the common electrophoretically slow form (C3S) found in BALB/c mice and most other mouse strains. Furthermore, studies on the decay rate of C3 revealed that C3b/iC3b **fragments** are converted to C3c/d at a faster rate in sera from female SJL/J mice compared to female BALB/c mice. Because removal and solubility of immune complexes is influenced by complement C3, it is possible that the more rapid decay of the phagocytic ligands C3b/iC3b may account for the increased susceptibility to development of autoimmune disease in female SJL/J mice.

Tags: Animal; Comparative Study; Female; Male; Support, Non-U.S. Gov't

Descriptors: *Complement 3--chemistry--CH; Autoimmune Diseases--genetics --GE; Autoimmune Diseases--immunology--IM; Complement 3--genetics--GE; Complement 3--metabolism--ME; Complement 3b--metabolism--ME; Complement Activation; Immunogenetics; Mice; Mice, Inbred BALB C; Molecular Structure; Myositis--genetics--GE; Myositis--immunology--IM; Phagocytosis; Polymorphi sm (Genetics); Species Specificity

CAS Registry No.: 0 (Complement 3); 80295-43-8 (Complement 3b)

Record Date Created: 19930408

Record Date Completed: 19930408

07878296 93339113 PMID: 8339607

Genetic markers for chronic bronchitis and peak expiratory flow in the Copenhagen Male Study.

Vestbo J; Hein H O; Suadicani P; Sorensen H; Gyntelberg F
Department of Occupational Medicine, Rigshospitalet, Copenhagen.

Danish medical bulletin (DENMARK) Jun 1993, 40 (3) p378-80, ISSN 0907-8916 Journal Code: 0066040
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

The associations between four major blood groups, ABH secretor status, and complement C3, and chronic bronchitis and peak expiratory flow were examined in 3387 men, aged 55-74 years. Presence of chronic bronchitis was assessed using the British Medical Research Council (BMRC) questionnaire. Men with NS- in the MNS system had significantly less chronic bronchitis than others, i.e. 11.4% versus **16** .0% ($p < 0.0005$). In heavy smokers, both men with NS- and NS+ had significantly less chronic bronchitis than others. Also, Rhesus positive men had less chronic bronchitis than Rhesus negative men, 28.0% versus 38.3% ($p < 0.05$). Absence of the complement C3 allele **C3F** was associated with a higher prevalence of chronic bronchitis. No association with chronic bronchitis was found for the AB0 system or the ABH secretor system. No convincing associations were found between genetic markers and mean peak flow. In conclusion, this study has suggested new genetic markers for respiratory disease indicating a partly genetic aetiology of chronic respiratory disease. The MNS, and to some extent the Rhesus blood groups, were associated with chronic bronchitis in middle-aged men. However, this study demonstrates once again that no single blood type can act as a risk indicator of chronic respiratory disease.

Tags: Human; Male; Support, Non-U.S. Gov't
Descriptors: *Blood Groups; *Bronchitis--blood--BL; *Bronchitis

08105071 94170831 PMID: 8125130

C3 binds with similar efficiency to Fab and Fc regions of IgG immune aggregates.

Anton L C; Ruiz S; Barrio E; Marques G; Sanchez A; Vivanco F
Department of Immunology, Fundacion Jimenez Diaz, Madrid, Spain.

European journal of immunology (GERMANY) Mar 1994, 24 (3) p599-604, ISSN 0014-2980 Journal Code: 1273201
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

The covalent binding reaction of the third component of complement (C3) with rabbit IgG immune aggregates has been studied by enzymic digestion of C3b-IgG adducts. In these adducts C3b was radioactively labeled in the free thiol group generated during activation of the internal thioester of C3. Trypsin digestion of 14C-labeled C3b-IgG adducts degrades C3b to a small antibody-bound 14C-labeled C3 **fragment** (14C- **C3frg**), whereas the antibody remains unaltered. Papain digestion of trypsin-treated 14C- **C3frg** -IgG complexes generated Fc and Fab **fragments** bearing equivalent amounts of covalently bound 14C- **C3frg** (43% and 40%, of the total C3 present in the aggregates, respectively). Hydroxylamine treatment of the 14C- **C3frg** -Fab and 14C- **C3frg** -Fc complexes released a 14C- **C3frg** of similar size (about 3-4 kDa) in which the N-terminal residue was the radiolabeled Cys1010. A **fragment** with the same radioactive N terminus and characteristics was obtained by sequential trypsin and papain digestion of purified C3 labeled with iodo-[14C] acetamide. Affinity-purified 14C- **C3frg** -Fc complexes digested with pepsin generated a mixture of radioactive **peptides** , most probably complexes formed by 14C- **C3frg** and C gamma 2 or the hinge digestion products, and 14C- **C3frg** -pFc' complexes. The latter was also immunoprecipitated with anti-Fc-Sepharose from the pepsin digestion supernatants of 14C-labeled-C3b-IgG complexes. Taken together these data indicate that, during complement activation through the alternative pathway by IgG immune aggregates, C3 is not bound to a single site on the antibody molecule. Both Fab and Fc regions of IgG are equally efficient targets for C3 anchorage. In addition, the data confirm the pFc' as a region of C3 attachment within the Fc **portion** , and strongly suggest that C3b is bound either to the C gamma 2 domain or the hinge or both.

Tags: Animal; In Vitro; Support, Non-U.S. Gov't

08878431 20164388 PMID: 10699425

Assay of a seric human hexapeptide (HWESAS) using a monoclonal antibody and ELISA.

Capiaumont J; Jacob C; Sarem M; Nabet P; Belleville F; Dousset B

Laboratory of Medical Biochemistry, School of Medicine, Henri Poincare Nancy I University, P.O. Box 184, 54505, Vandoeuvre-les-Nancy, France.

Clinica chimica acta; international journal of clinical chemistry (NETHERLANDS) Mar 2000, 293 (1-2) p89-103, ISSN 0009-8981
Journal Code: 1302422

Document type: Clinical Trial; Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Human serum contains low-molecular-weight growth factors potentiating some in vitro biological effects of IGF-I and IGF-II and recently two **peptides** were mainly identified: HWESAS and WGHE. In order to determine seric HWESAS concentration, a specific monoclonal antibody against HWESAS was prepared. Its specificity was studied by inhibition tests: this antibody cross-reacts with Y-HWESAS, Cys-HWESAS. It does not react with HWESAS when its COOH is blocked, or with HWE, WGHE and tryptophan or with **C3f** (SSKITHRIHWESASLLR) which is a **fragment** of human complement containing HWESAS motif. Its affinity was measured by non competitive enzyme immunoassay (3.89+/-2.44.10(8) M(-1)). Then, this antibody was used in enzyme-linked immunosorbent assay (ELISA) and the preliminary assays were performed to detect HWESAS in serum. In contrast to healthy subjects, patients with chronic renal failure exhibited undetectable concentration of hexapeptide while after successful renal transplantation values increased to reach levels found in healthy subjects and varying according to post-operative evolution. These data are a strong hint that the kidney plays an important role in the production of this hexapeptide and underly the clinical interest of HWESAS detection in renal pathology.

Tags: Animal; Human; Support, Non-U.S. Gov't

Descriptors: *Antibodies, Monoclonal--diagnostic use--DU; *Oligopeptides --analysis--AN; Amino Acid Sequence; Antibodies, Mo

09584636 21368159 PMID: 11473682

Complement analysis in children with idiopathic membranoproliferative glomerulonephritis: a long-term follow-up.

Schwertz R; Rother U; Anders D; Gretz N; Scharer K; Kirschfink M

Institute of Immunology, University of Heidelberg, Im Neuenheimer Feld 150, D-69120 Heidelberg, Germany. RainerSchwertz@ukl.uni-heidelburg.de

Pediatric allergy and immunology - official publication of the European Society of Pediatric Allergy and Immunology (Denmark) Jun 2001, 12 (3) p166-72, ISSN 0905-6157 Journal Code: 9106718

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Fifty children with idiopathic membranoproliferative glomerulonephritis (MPGN), aged 2-14 years at apparent onset, were monitored for the presence of C3 nephritic factor (C3 NeF) and signs of complement activation in serum. In addition, C3 allotyping was performed in 32 patients. Observation time ranged from 2 to 20 (median 11) years. C3 NeF activity was detected at least once in 60% of the patients (in 11 of 26 with type I, in **15** of 17 with type II, and in four of seven with type III). C3 NeF-positive patients had significantly reduced levels of CH50 and C3 and elevated levels of C3dg/C3d. During follow-up, C3 levels were persistently normal in 62% of the patients with MPGN type I and in 43% with type III but in only 18% with type II. C3 allotype frequencies differed from those found in healthy controls with a significant shift to the **C3F / C3FS** variants in C3 NeF-positive patients. C3b(Bb)P as a marker for alternative pathway activation was not increased in C3 NeF-positive patients. Despite the presence of C3 NeF activity, C3 levels remained normal in six patients throughout the observation period. C3 NeF became undetectable in six patients, whereas seven developed C3 NeF activity during follow-up. There was no significant difference in renal survival probability in patients with or without C3 NeF activity. Neither C3 variants nor continuous low C3 or low CH50 levels had any prognostic value for the clinical outcome. No factor H deficiency was detected.

Tags: Comparative Study; Human; Male; Support, Non-U.S. Gov't

Descriptors: *Complement 3--analysis--AN; *Glomeruloneph

10976707 97329479 PMID: 9185962

Purification and characterization of bovine complement component C3 and its cleavage products.

Di Carlo A L; Paape M J; Hellman J; Lilius E M

Department of Animal Sciences, University of Maryland, College Park 20742, USA.

American journal of veterinary research (UNITED STATES) Jun 1997, 58 (6) p585-9, ISSN 0002-9645 Journal Code: 0375011

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

OBJECTIVE: To purify complement component C3 from bovine serum, characterize and analyze NH2-terminal amino acid sequences from its various cleavage products, and do cross-species homology comparisons. ANIMALS: 2 healthy lactating Holstein cows, and 2 healthy adult female New Zealand White rabbits. PROCEDURE: Bovine C3 was isolated from serum, and was cleaved to C3b. The resulting protein was analyzed to determine apparent molecular mass of resulting protein segments. Bands were electroblotted onto a membrane and excised, then NH2-terminal amino acid sequences were determined. RESULTS: The C3 preparation consisted of 6 segments, with molecular mass of 30, 40 (2 bands, a and b), 70, 75, and 115 kd. Via sequence comparisons, the 115-kd band was identified as the alpha chain; the 75-kd segment was determined to be the NH2-terminal **portion** of alpha chain; the 70-kd piece was identified as the intact beta chain; and the two 40-kd bands are believed to be located at the C-terminal **portion** of the alpha chain, at the cleavage site that yields **C3f** . The 30-kd band is the NH2-terminal **portion** of the alpha chain (minus the C3a segment). Sequence analysis of each band revealed a high degree of homology with human, rat, mouse, and horse C3. Polyclonal antibodies raised in rabbits yielded sera that reacted to the purified sample in manner similar to that of commercially available antibodies. CONCLUSIONS: The purified preparation contained intact C3, C3b, and the degradation products iC3b and C3c, which had high sequence homology with those of other species. The C3a and C3d, and C3g segments of protein were not detected and may have been lost during purification, lyophilization, or transfer steps. Structure and cleavage characteristics of bovine C3 can be used to better understand immune responses to bacterial pathogens in the mammary gland.

Tags: Animal; Female; Human

Descriptors: *Complement 3--chemistry--CH; *Complement 3--isolation and purification--IP; Amino Acid Sequence; Antibodies--immunology--IM; Cattle; Complement 3--immunology--IM; Electrophoresis, Polyacrylamide Gel --veterinary--VE; Horses; Immunodiffusion--veterinary--VE; Mice; Molecular Sequence Data; Rabbits; Rats; Sequence Homology, Amino Acid; Species Specificity

CAS Registry No.: 0 (Antibodies); 0 (Complement 3)

Record Date Created: 19970909

Record Date Completed: 19970909

1438276 98321171 PMID: 9647737

Purification from human plasma of a hexapeptide that potentiates the sulfation and mitogenic activities of insulin-like growth factors.

Dousset B; Straczek J; Maachi F; Nguyen D L; Jacob C; Capiaumont J; Nabet P; Belleville F

Laboratory of Biochemistry, Nancy Medical School, Henri Poincare-Nancy I University, Vandoeuvre, France.

Biochemical and biophysical research communications (UNITED STATES) Jun 29 1998, 247 (3) p587-91, ISSN 0006-291X Journal Code: 0372516

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The human plasma contains small **peptide** molecules known as low molecular weight growth factors synergistically increasing certain biological actions of insulin-like growth factors. In the present work we isolated and characterized a hexapeptide with HWESAS as structure. This purified **peptide** was absolutely necessary for the sulfation activity of insulin-like growth factor-I on chick embryo pelvic cartilages and improved the mitogenic activity of both insulin-like growth factors. The effects of this hexapeptide were confirmed by using the homologous synthetic **peptide** , that exhibited similar biological effects. Other synthetic **peptides** with structure derived from hexapeptide were shown to be active: the pentapeptide HWESA appeared more potent than the tripeptide HWE, which is about 170 to 200 times less active than the hexapeptide. The sequence of hexapeptide HWESAS is identified in only one human protein that is **C3f** , a **fragment** of C3 complement.

Tags: Animal; Human; Support, Non-U.S. Gov't

Descriptors: Mitogens--chemistry--CH; * **Peptides** --blood--BL; *Somatomedins--physiology--PH; *Sulfates--metabolism--ME; Cartilage--drug effects--DE; Cartilage--growth and development--GD; Chick Embryo; Complement 3--chemistry--CH; Growth Substances--blood--BL; Insulin-Like Growth Factor I--physiology--PH; **Peptide Fragments** --chemistry--CH; **Peptide Fragments** --pharmacology--PD; **Peptides** --physiology--PH; Sequence Analysis; Spectrum Analysis, Mass

CAS Registry No.: 0 (Complement 3); 0 (Growth Substances); 0 (Mitogens); 0 (Peptide Fragments); 0 (Peptides); 0 (Somatomedins); 0 (Sulfates); 0 (complement 3f); 67763-96-6 (Insulin-Like Growth Factor I)

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Processing of serum proteins underlies the mass spectral fingerprinting of myocardial infarction.

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The MALDI-TOF spectra of **peptides** from the sera of normal and myocardial infarction patients produced patterns that provided an accurate diagnostic of MI. In myocardial infarction, the spectral pattern originated from the cleavage of complement C3 alpha chain to release the **C3f peptide** and cleavage of fibrinogen to release **peptide** A. The fibrinogen **peptide** A and complement **C3f peptide** were in turn progressively truncated by aminopeptidases to produce two families of **fragments** that formed the characteristic spectral pattern of MI. Time course and inhibitor studies demonstrated that the **peptide** patterns in the serum reflect the balance of disease-specific-protease and aminopeptidase activity ex vivo.

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